

**Patent claims:**

1. Method for detecting the effect of different chemotherapeutic agents and/or radiation therapy in malignant diseases, wherein the expression profiles of apoptosis-regulating and/or cell growth regulating genes and/or individual differences (mutations) in the gene sequences is determined and the changes associated with chemotherapeutic agents and/or radiation therapy are identified, represented and diagnostically evaluated.
2. Method in accordance with claim 1, wherein the expression profiles of the genes of the Bcl-2 family, preferably Bax, p53, p16, caspases, Rb, cyclins, inhibitors of cyclin-dependent kinases (CDKIs), ATM and inhibitors of apoptosis proteins (IAPs) and/or mutations in the genes are determined using protein or DNA/RNA analyses and evaluated singly or in various combinations.
3. Method in accordance with claim 1 or 2, wherein individual differences in the sequence of apoptosis and/or cell growth-regulating genes and or the expression of their gene products, which occur in malignant diseases, are related to an individually different responsiveness to drugs and are evaluated, particularly with regard to their relevance to the response to therapy.
4. Method for selecting more efficacious therapeutic agents for the treatment of malignant diseases, wherein the status of cell cycle genes and/or of apoptosis-associated target genes or of their gene products in body fluids, cells or organs are determined ex vivo and the more efficacious agents for this status are selected.
5. Method in accordance with claim 4, wherein agents for the treatment of leukemic diseases and other hematological

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malignomas and solid tumors like, for example, tumors of the gastrointestinal tract, pancreas, prostate, gynecological tumors, sarcomas, brain tumors, skin and lung tumors as well as tumors of endocrine organs are evaluated.

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6. Method in accordance with claim 4 or 5, wherein therapeutic agents are known cytostatic agents, preferably steroid hormones, alkylating agents, anthracyclines, antimetabolites, taxanes, topoisomerase inhibitors, Vinca alkaloids, cisplatin and other platinum analogues and many more.
7. Method in accordance with claim 1 to 6, wherein for the treatment of solid tumors or leukemic or other hematological malignant diseases, preferably of chronic lymphocytic leukemia, the Bax expression or mutations are evaluated and, with a low Bax expression, a treatment with alkylating agents, anthracyclines and Vinca alkaloids is avoided and another form of therapy is chosen.
8. Method in accordance with claim 1 to 6, wherein for the treatment of leukemic diseases, mainly of chronic lymphocytic leukemia, the Bax expression or mutations are evaluated and, with low Bax expression, a treatment with steroid hormones or fludarabine phosphate (2-CDA) is carried out.
9. Method in accordance with claim 1 to 6, wherein for the treatment of leukemic diseases, mainly of chronic lymphocytic leukemia, the p53 expression or mutations are evaluated and, with the presence of mutations within the coding sequence regions of the p53 genes, a treatment with DNA-damaging substances, particularly with alkylating agents, anthracyclines and fludarabine, is avoided and another form of therapy is selected.
10. Method in accordance with claim 1 to 9, wherein by combination of the determination of the status of different

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apoptosis and/or cell growth-associated genes, mainly of p53 and Bax or their gene products and/or mutations and/or their homologues, individual schemes of treatment are drawn up.

11. Use of the status determination via protein or DNA/RNA analyses of apoptosis and/or cell growth-associated genes or their gene products for determination of resistances to radiation therapy and to therapeutic agents as well as for the purposeful selection of therapeutic agents for cytotoxic therapies.
12. Use in accordance with claim 11, wherein the status, namely the expression profile and/or mutations of genes of the Bcl-2 family, mainly Bax, p53, p16, caspases, Rb, cyclins, inhibitors of the cyclin-dependent kinases (CDKIs), ATM and inhibitors of the apoptosis proteins (IAPs), of individual genes or in different combinations is determined.
13. Use in accordance with claim 12, wherein the status of Bax expression or mutations of the Bax gene or Bax promoters and Bax regulators is used.
14. Use in accordance with claim 12, wherein the status of the p53 expression or mutations of the p53 gene is used.
15. Use in accordance with claim 11 to 14, wherein the status of Bax and p53 risk-adapted tumor therapies with malignant hematological diseases, like CLL, and other tumoral diseases is used.
16. Use in accordance with claim 11 to 15 of a combination of p53 and Bax and possibly other cell cycle and apoptosis regulators either alone or in their combination in the sense of a molecular pathway diagnosis with malignant tumors or precancers.